CARDIAC MAGNETIC RESONANCE ASSESSMENT OF PROGRESSIVE MYOPERICARDITIS DUE TO COBALT CARDIOTOXICITY

Massimiliano Lorenzini$^{1,2}$, Mohammed Y. Khanji$^{1,3}$, Luis Rocha Lopes$^{1,2}$, Charlotte Manisty$^{1,2}$, Konstantinos Savvatis$^{1,2,3}$.

1 Department of Cardiovascular Imaging, Barts Heart Centre, St. Bartholomew’s Hospital, London, UK;
2 University College London Institute of Cardiovascular Science, London, UK;
3 William Harvey Research Institute, NIHR Barts Biomedical Research Centre, Queen Mary University London, London, UK.

Conflicts of Interest: Nothing to Disclose.

**Corresponding author:** Dr. Massimiliano Lorenzini
Inherited Cardiovascular Diseases, Barts Heart Centre
St Bartholomew’s Hospital
W Smithfield, EC1A 7BE
London, United Kingdom
dr.m.lorenzini@gmail.com
A 48-year-old man was referred with a 3-year history of recurrent pericardial effusion requiring drainage, and progressive heart failure. Past medical history was unremarkable other than bilateral hip replacement with metal-on-metal implants for arthritis at 39-years of age.

ECG showed low voltages (panel B). Bloods, including an extensive rheumatology work up, were unremarkable apart from persistently mildly elevated troponin T (40ng/l, normal <14ng/l). Initial cardiac magnetic resonance (CMR) showed biventricular dysfunction with subepicardial infero-lateral and midwall septal late enhancement (LGE, panel A). FDG-PET showed mild diffuse cardiac uptake and corresponding to the hip prostheses (panel H). Endomyocardial biopsy showed myocyte hypertrophy, focal fibrosis and endocardial thickening with no evidence of active myocarditis or amyloid. Repeat CMR at 15 months showed chronic pericardial effusion, a now severe biventricular dysfunction (Panel C, Supplementary Video 1), mildly raised native T2 (55ms @ 1.5T, normal 40-51ms, panel D) and native T1 (1080ms, normal 970-1050ms, panel E), left ventricular apical thrombus on early gadolinium enhancement (panel F, arrow) and a striking progression of LGE (panel G). Myocardial T2* was normal (45ms) and liver T2* was mildly reduced (10ms).

Serum chromium and cobalt levels were extremely elevated [1279 nmol/L (normal<134 nmol/L) and 5647 nmol/L (normal<118 nmol/L), respectively] and subsequent mass spectrometry of the myocardial biopsy confirmed very high myocardial levels [17.2 ug/g (normal<0.6 ug/g) and 4.7 ug/g (normal<0.5 ug/g), respectively].

CMR can demonstrate the typical features of this rare but recognised complication of metal-on-metal hip prostheses, in this case with a striking progression of LGE.